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Chapter

Dental Biofilm as Etiological Agent of Canine Periodontal Disease

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Abstract

Periodontal disease is one of the most common health problem affecting dogs. The disease is more prevalent in small breeds and brachycephalic breeds compared to large breeds, and incidence increases with advancing age. In first stage it affects only the gingival tissue and causes gingivitis. It later develops into periodontitis which involves changes in other periodontium tissues. Main etiological agents of periodontal disease are pathogenic bacteria of dental biofilm, and products of their metabolism. In human, *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia* play a key role in the etiology of periodontal disease. Also, there are many other candidates as human periodontal pathogens, including *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *Prevotella nigrescens*, *Fusobacterium nucleatum*, *Parvimonas micra*, *Eikenella corrodens*, *Capnocytophaga gingivalis*, *Eubacterium nodatum* and *Campylobacter rectus*. Since periodontal diseases in dogs are similar to human diseases in terms of disease progression and clinical manifestation, we can assume their common etiology. This chapter is focused on review about canine dental biofilm and about members of biofilm as potential causative agent of canine periodontal disease.

Keywords: dental plaque, biofilm, dog, periodontal disease, gingivitis, periodontitis

1. Introduction

Periodontal disease is a significant veterinary health problem of companion dogs [1]. Periodontal disease refers to a group of inflammatory diseases. In both humans and dogs the initial stages of periodontal disease are observed clinically as red and inflamed gingivae, defined as gingivitis. Without treatment to remove, and disrupt the dental plaque, gingivitis may progress to periodontitis [2]. Periodontitis, the later, irreversible stage of the disease, is an inflammatory disease of supporting teeth tissues [3]. The primary etiological factor in the initiation and progression of periodontal disease is dental plaque [4]. Dental plaque is the community of microorganisms found on a tooth surface as a biofilm, embedded in a matrix of polymers of host and bacterial origin [5]. It is believed that enzymes secreted by dental biofilm bacteria as well as bacterial antigens activate the host inflammatory response initiating disease [6]. Dental calculus that represents mineralized bacterial dental biofilm is considered as secondary etiological factor in periodontal disease [7]. Dental calculus itself is relatively non-pathogenic and, despite its rough surface, is not the direct cause of inflammatory processes, but mainly has an irritant effect.

In addition, the presence of dental calculus leads to greater biofilm accumulation by creating a rough surface [8]. Dental calculus is always covered with a layer of dental biofilm, so it plays an important role as retention factor in the colonization of microorganisms [9].

2. Canine dental biofilm

The oral cavity is a host for a variety of microorganisms including bacteria, viruses, fungi and protozoa that colonize teeth, tongue, oral mucosa, hard palate, caries lesions, periodontal pocket and similarly. The distribution of microorganisms in the oral cavity is not random; most species prefer certain places to others due to the specific local conditions that these sites provide, for example, the anaerobic environment of the gingival sulcus [10, 11]. However, the oral cavity environment is also hostile to microbial life, so only a certain groups of microorganisms entering it are able to colonize it, and survive in this environment. Microorganisms must attach to the surface and form biofilms to remain in oral cavity [12].

Dog oral cavity hides a rich and diverse bacterial community and exceeds the estimates of culture-based studies. Of the cultivable oral microbiota, genera *Actinomyces*, *Streptococcus* and *Granulicatella* are most commonly isolated from saliva. Genera *Porphyromonas*, *Actinomyces* and *Neisseria* are most commonly isolated from plaque [13]. Genera *Porphyromonas*, *Fusobacterium*, *Capnocytophaga*, *Derxia*, *Moraxella*, *Bergeyella*, non-cultivable *Lachnospiraceae*, *Enhydrobacter*, non-classified *Peptostreptococcaceae*, *Xylanibacter*, *Parabacteroides*, *Tannerella*, *Neisseria*, *Treponema* and *Bacteroides* were identified by the pyrosequencing of the 16S rRNA gene [14]. In another oral microbiota study also by pyrosequencing the 16S rRNA gene, the bacterial genera *Actinomyces*, *Porphyromonas*, *Fusobacterium*, *Neisseria*, *Pasteurella*, *Lampropedia*, *Capnocytophaga*, *Frigovirgula*, *Filifactor*, *Conchiformibius*, *Eubacterium*, *Streptococcus*, *Corynebacterium* and *Derxia* have been identified with an abundance >1% [15]. Based on the sequencing of the 16S rRNA gene the presence of other genera in the oral cavity of dogs such as *Abiotrophia*, *Aerococcus*, *Campylobacter*, *Cardiobacterium*, *Clostridium*, *Curtobacterium*, *Dialister*, *Dietzia*, *Dysgonomonas*, *Eikenella*, *Enterococcus*, *Eubacterium*, *Gemella*, *Globicatella*, *Granulicatella*, *Haemophilus*, *Lactobacillus*, *Leptotrichia*, *Leucobacter*, *Micrococcus*, *Micromonas*, *Peptostreptococcus*, *Prevotella*, *Propionibacterium*, *Propionivibrio*, *Rothia*, *Selenomonas*, *Schwartzia*, *Sporocytophaga*, *Wolinella*, *Xanthomonas* and *Xenophilus* was confirmed [16]. Many of them are part of the biofilm formed on teeth surface. In dogs, also in humans, a subgingival biofilm includes colonies of anaerobic, Gram-negative (*Bacteroides* spp., *Capnocytophaga* spp., *Fusobacterium* spp., *Porphyromonas* spp., *Prevotella* spp., *Tannerella* spp. and *Treponema* spp.) as well as Gram-positive bacteria (*Actinomyces* spp., *Corynebacterium* spp., *Eubacterium* spp., *Peptostreptococcus* spp. and *Streptococcus* spp.) [17].

The formation of dental biofilm in the oral cavity is a multi-stage process [18]. It can be divided into four main stages: pellicle formation, initial bacterial adhesion, plaque maturation and finally bacterial dispersion [11]. Initially, a semipermeable layer called pellicle is formed on the tooth surface, which mediates the interaction between tooth, oral fluids and microorganisms [19]. Primary colonizers form biofilm autoaggregation (aggregation between the same species) and coaggregation (aggregation between different species) [20]. In addition, they facilitate the arrival of additional bacteria by providing multiple diverse adhesive sites. They also begin to build a matrix that holds the biofilm together. Some species are incapable of adhering to the surface, but are often able to anchor to a matrix or directly to earlier colonizers [21]. Representatives of the genera *Neisseria*, *Corynebacterium*

and *Stenotrophomonas* are involved as primary colonizers in the formation of canine dental biofilm. The most common species of the genus *Neisseria* are *N. zoodegmatis*, *N. animaloris* and *N. weaveri*. Representatives of the genera *Actinomyces*, *Porphyromonas*, *Moraxella*, *Leucobacter*, and the families *Peptostreptococcaceae* and *Pasteurellaceae* probably play the roles of secondary colonizers. Species *Actinomyces canis* and *Porphyromonas gingivicanis* show high levels of biofilm incorporation. The species which featured most frequently in the role of third community member are *Peptostreptococcaceae* spp., *Porphyromonas gingivicanis* and *Leucobacter* spp. [22]. Bacteria from dental biofilm can either be protective, and provide an essential barrier through interactions with the host immune system, or be pathogenic, and cause diseases, such as periodontitis [15]. Oral microbiota varies greatly in healthy dogs and in dogs with disease of oral cavity, and also contains a high proportion of non-cultivable or unexplored species [23]. In healthy dogs, more common species are *Moraxella* spp., *Bergeyella zoohelcum*, *Neisseria shayeganii*, *Pasteurellaceae* spp., *Capnocytophaga* spp. and *Stenotrophomonas* spp. In dogs with periodontitis, species *Peptostreptococcaceae* spp., *Lachnospiraceae* spp. and *Clostridiales* spp. are significantly more prevalent [24].

3. Periodontal disease

Periodontal disease occurs naturally in a wide range of species from rodents to humans [25]. Periodontal disease is one of the most common diseases of adult dogs, with up to 80% of animals affected [23]. All canine breeds are at risk of developing periodontal disease [26]. In general, the disease is more prevalent in small breeds compared to large breeds, and incidence increases with advancing age. In addition, brachycephalic breeds and dogs with teeth overcrowding have been reported to be especially vulnerable to developing the advanced stages of the disease [27]. There are four stages of periodontal disease, each of which is based on the severity of clinical lesions as follows: Stage 1—gingivitis, Stage 2—early periodontitis, Stage 3—moderate periodontitis, Stage 4—advanced periodontitis [28].

Gingivitis is completely reversible, and is recognized by the classic signs of halitosis, bleeding, inflammation, redness and swelling of the gingivae. Periodontitis is irreversible, and attacks the deeper structures that support the teeth, permanently damaging the surrounding bone and periodontal ligament [23]. The breakdown of the collagen fibers of the periodontal ligament results in a periodontal pocket between the gingiva and the tooth. Periodontal pocket deepen due to further destruction of periodontal ligament fibers and alveolar bone resorption. Advanced periodontitis is characterized by gingival erythema and edema, gingival bleeding, gingival recession, tooth mobility, suppuration of periodontal pocket and loss of teeth [29]. We know two main categories of periodontal disease in which loss of supporting structures around the tooth occurs: chronic periodontitis and aggressive periodontitis [30]. Chronic periodontitis is chronic inflammation results in, mostly irreversible, loss of epithelial tissue, bone and ligament. Aggressive periodontitis is characterized by rapid rate of disease progression. It can be present in localized or generalized form; both are early-onset forms of chronic periodontal inflammatory disease. No disease-specific biomarkers exist that differentiate chronic periodontitis from aggressive periodontitis. Although current knowledge suggests that both have similar etiology and histopathology and might indeed be different ends of the same disease spectrum [31].

Periodontal disease is caused by the accumulation of bacterial dental biofilm on the teeth and gingivae, toxic products of the metabolism of these microorganisms, and the host immune response against the infection that triggers the inflammatory

process [32]. In case of chronic periodontitis usually have abundance of plaque and calculus, which match with the amount of periodontal destruction. On the other hand, in case of aggressive periodontitis, there is usually a mismatch between the amount of local factors and the periodontal destruction [33]. Periodontal disease affects more frequently and more severely regions of premolars and molars than regions of maxillary and mandibular incisors. Missing of teeth is observed at a high and increasing incidence with age. The tooth most commonly lost is the first premolar, followed by the other premolars and molars, where severe periodontitis is frequently found [34]. Periodontitis is a serious infection that can have medical consequences such as anorexia and weight loss, chronic pain, swollen gums, dental caries, breakage or loss of teeth and breakage of the maxillary or mandibular bone [35]. Unfortunately, the damage from periodontal disease is not confined to just loss of teeth. Oral infection, especially periodontitis, may affect the course and pathogenesis of a number of systemic diseases, such as chronic bronchitis, pulmonary fibrosis, endocarditis, interstitial nephritis, glomerulonephritis and hepatitis [1].

4. Periodontal pathogens

Although there is sufficient evidence that biofilm accumulation and maturation is essential for initiation and progression of periodontal disease, studies show that bacterial species colonizing periodontal pocket have different roles in the pathogenesis of this disease [36]. Microbial density is considered to be critical for the development of gingivitis, and some types of chronic periodontitis, while the species of the microorganisms may be of greater importance in the initiation of aggressive periodontitis [35]. Subgingival microbiota in periodontitis may contain hundreds of bacterial species, but only a small number is associated with disease progression, and is considered to be of importance etiologically [37]. The presence of *Mycobacterium tuberculosis* is an indication of tuberculosis, and *Treponema pallidum* a positive diagnosis of syphilis, but there is no single microorganism, which is attributable to chronic periodontitis [38].

As with any other infection, identification of the microbial pathogens associated with the etiology of periodontitis is the first step towards the development of effective therapeutic approaches. The establishment of a microorganism as a true pathogen should be based on two main levels of evidence: (1) the organism should be present in higher prevalence and/or levels in disease than in health, and (2) its suppression or elimination should reduce or stop disease progression [39]. In human, the presence of three species of Gram-negative anaerobic bacteria within subgingival biofilm, *Porphyromonas gingivalis*, *Treponema denticola* and *Tannerella forsythia*, described by Socransky et al. as the "red complex," show a strong association to periodontitis, and some studies have indicated their involvement also in dogs [40]. There are many others candidates as human periodontal pathogens, including *Aggregatibacter actinomycetemcomitans*, *Prevotella intermedia*, *Prevotella nigrescens*, *Fusobacterium nucleatum*, *Parvimonas micra*, *Eikenella corrodens*, *Capnocytophaga gingivalis*, *Treponema socranskii*, *Eubacterium nodatum* and *Campylobacter rectus* [41]. Some of them are also associated with canine periodontal disease. *Tannerella forsythia*, *Campylobacter rectus* and *Porphyromonas gulae* were detected in almost all dogs with periodontitis. *Prevotella intermedia* and *Eikenella corrodens* were detected only in few dogs with periodontitis, *Treponema denticola*, *Capnocytophaga ochracea* and *Porphyromonas gingivalis* were detected in only one specimen. In addition, *Prevotella nigrescens* and *Aggregatibacter actinomycetemcomitans* were not detected in any of the specimens [42]. *Actinomyces canis* and *Corynebacterium canis* are significantly more prevalent in dogs with periodontitis than in healthy dogs [23, 24].

Species of the genera *Actinomyces* and *Corynebacterium* can play the same role in periodontitis in dogs that *P. gingivalis* plays in periodontitis in humans. This assumption is based on the finding that the ratio of these genera that have trypsin-like activity is increased in areas with periodontitis compared to their ratio in non-periodontal areas and may partly explain the absence of *P. gingivalis* [43]. Protist organisms, such as *Entamoeba gingivalis* and *Trichomonas tenax*, are also present in canine dental biofilm, and correlate with periodontal disease. These findings provide the evidence for the presence of oral protozoa in canine dental biofilm, and suggest a possible role for protozoa in the periodontal disease process [44].

4.1 Genus *Porphyromonas*

The genus *Porphyromonas* is phylogenetically classified in the family *Porphyromonadaceae*, order *Bacteroidales*, class *Bacteroides* and phylum *Bacteroidetes*. Representatives of this genus are Gram-negative, obligate anaerobic, non-motile and non-spore-forming rods. Several species of *Porphyromonas*, including *P. endodontalis*, *P. circumdentaria* and *P. gingivalis* were isolated from the biofilm of adult dogs, but not from any oral sites of puppies and adolescent dogs [45]. Also, several new *Porphyromonas* species (*P. gulae*, *P. macacae*, *P. cangingivalis*, *P. cansulci*, *P. creviocanis*, *P. gingivacanis*, *P. canoris*, *P. denticanis*) associated with periodontal disease have been described [23]. In humans, the major periodontal pathogen is *P. gingivalis* [46]. There are several differences between isolates *Porphyromonas* from humans and from dogs. For example, *P. gingivalis* isolates of canine origin are catalase positive, but isolates of human origin are catalase negative. These catalase positive organisms like *P. gingivalis* may represent the species *P. gulae* [47].

P. gulae is rarely found in humans and healthy animals, usually is isolated from animals, including dogs, with active periodontitis [48]. From dogs with periodontal disease are most frequently isolated three *Porphyromonas* species: *P. gulae*, *P. salivosa* (current name, *P. macacae*) and *P. denticanis* [49]. Of these only, *P. gulae* exhibits virulence characteristics similar to those of the human periodontal pathogen *P. gingivalis* such as lysyl- and arginyl-specific proteolytic activity of the gingipains. This finding suggested that *P. gulae* may play a key role in the development of periodontitis in dogs [50]. In addition, fimbrial protein with the same size and antigenicity similar the 41-kDa fimbrial subunit protein (fimbrillin, FimA) of *P. gingivalis* was identified in *P. gulae* [51]. The fimbria is an important cell structure involved in the adherence and invasion of host's cells, and stimulates the production of inflammatory cytokines by macrophages and fibroblasts. This adhesive ability is considered to be a major pathogenic characteristic of *Porphyromonas* that causes periodontal tissue destruction [52].

P. cangingivalis is the most prevalent canine oral bacterial species in both plaque from healthy gingiva and plaque from dogs with early periodontitis. The ability of *P. cangingivalis* to predominate in both health and disease environments suggests that it is both metabolically flexible enough to colonize in health and also able to compete against other *Porphyromonas* spp. in a disease environment [24]. *P. cangingivalis* has a complete protoporphyrin IX synthesis pathway potentially allowing it to synthesize its own heme unlike pathogenic Porphyromonads such as *P. gingivalis* that acquire heme predominantly from blood. The ability to synthesize siroheme and vitamin B_{12} point to enhanced metabolic flexibility for *P. cangingivalis*, which may underlie its prevalence in the canine oral cavity [53].

4.2 Genus *Tannerella*

The genus *Tannerella* is phylogenetically classified in the family *Porphyromonadaceae*, order *Bacteroidales*, class *Bacteroides* and phylum *Bacteroidetes*.

Representatives of this genus are Gram-negative, anaerobic, non-motile rods. The primary periodontal pathogen is *Tannerella forsythia* originally described as *Bacteroides forsythus*, and reclassified to *Tannerella forsythia* based on 16S rRNA phylogenetic analysis [54]. *T. forsythia* should be regarded as common member of oral microbiota in dogs [42], but dogs with gingivitis or periodontitis are more likely to be infected with *T. forsythia* than healthy animals [26]. *T. forsythia* has several virulence factors, including surface antigen BspA, cell surface proteolytic enzymes, hemagglutinin, cell envelope lipoproteins, glycosidases and cell surface (S)-layer, which contribute to pathogenic potential. The surface protein BspA can bind extracellular matrix components as well as other oral bacteria, and is partly responsible for alveolar bone resorption [55].

4.3 Genus *Campylobacter*

The genus *Campylobacter* is phylogenetically classified in the family Campylobacteraceae, order *Campylobacterales*, class *Epsilonproteobacteria* and phylum *Proteobacteria*. Species *Campylobacter rectus* (formerly *Wolinella recta*), Gram-negative, microaerophilic and motile bacterium, is regarded as common member of oral microbiota in dogs. *C. rectus*, *Tannerella forsythia* and *Porphyromonas gulae* are three major species present in dogs with periodontitis base on study using molecular biological approaches [42]. Several possible factors of virulence have been described for *C. rectus*, such as flagellum, surface layer proteins (S-layer), RTX-type toxins, GroELlike proteins and lipopolysaccharide [56]. *C. rectus* may be an important indicator of periodontal disease. Together with other oral anaerobic bacteria, *C. rectus* is associated with the initiation and progression of periodontal disease [57].

4.4 Genus *Treponema*

The genus *Treponema* is phylogenetically classified in the family *Spirochaetaceae*, order *Spirochaetales*, class *Spirochaetesa* and phylum *Spirochaetes*. *Treponemes* are Gram-negative, obligate anaerobic, motile spirochetes. *Treponemes* are involved in the development of chronic domestic animal diseases, including periodontal diseases in dogs [58]. Dogs harbor several different *Treponema* spp. in their oral cavity, and they can be common in both healthy and periodontitis affected dogs, indicating they are part of the normal oral microbiota [40]. Canine dental biofilm include species *T. denticola*, *T. socranskii*, *T. vincentii*, *T. maltophilum*, *T. medium* and *T. pectinovorum* [59]. Prevalence *T. denticola* and *T. socranskii* is significantly higher in dogs with periodontitis than in dogs without periodontitis. In addition, *Treponema* spp. are not only in the microbial biofilm but also within the gingival tissue [60].

Treponemes, including *T. denticola*, are found on the surface of dense subgingival bacterial biofilms, particularly at the interface of biofilms and gingival epithelium. *T. denticola* has been shown to adhere to fibroblasts and epithelial cells as well as extracellular components of the matrix present in periodontal tissues, and produces several harmful factors that can contribute to virulence of bacteria [61]. The main virulence factors of *T. denticola* in chronic periodontitis include motility and chemotaxis, the ability to interact synergistically with other periodontal pathogens, the ability to produce cytotoxic metabolites, the ability to form biofilms and a variety of cell surface proteins. Motility and chemotaxis allow the bacterium to rapidly colonize new sites, penetrate deep periodontal pocket and penetrate into epithelial layers. Cell surface proteins cause dysregulation of host defense, thereby helping to protect the subgingival biofilm and causing host tissue destruction [58].

4.5 Genus *Fusobacterium*

The genus *Fusobacterium* is phylogenetically classified in the family *Fusobacteriaceae*, order *Fusobacteriales*, class *Fusobacteriia* and phylum *Fusobacteria*. Species *Fusobacterium nucleatum* and *Fusobacterium canifelinum* were identified in subgingival plaque from dogs with and without periodontitis [62]. Based on phenotypic and genotypic differences, *F. nucleatum* is divided into five subspecies, namely *F. nucleatum* subspp. *nucleatum*, *F. nucleatum* subspp. *polymorphum*, *F. nucleatum* subspp. *fusiforme*, *F. nucleatum* subspp. *vincentii* and *F. nucleatum* subspp. *animalis*, whose prevalence varies with disease [63]. At present, the mechanisms of pathogenicity of *F. nucleatum* are unclear. Butyrate production is considered a virulence factor. The association of *F. nucleatum* with periodontal disease is probably through its role as a transient colonizer between Gram-positive and Gram-negative species, mainly in humans. Consequently, *F. nucleatum* can serve as a bridge between species that can colonize exposed tooth surfaces (early colonizers), and species that require interactions with other species (late colonizers). Since late colonizers tend to be species associated with periodontal destruction, bridging with *F. nucleatum* could play an important role in determining the pathogenicity of a mature oral biofilm community [64, 65]. Of the large number of periodontal pathogens, *F. nucleatum* is most frequently involved in infections outside the oral cavity, including pneumonia, pyogenic liver abscess, sepsis, infectious endocarditis, brain abscesses and caecal inflammation [66].

4.6 Genus *Parvimonas*

The genus *Parvimonas* is phylogenetically classified in the family *Peptoniphilaceae*, order *Tissierelliales*, class *Tissierellia* and phylum *Firmicutes*. The species *Parvimonas micra* originally classified as *Peptostreptococcus micros* was first reclassified in 1999 to *Mircomonas micros*, and the second time reclassified in 2006 to *Parvimonas micra* [67]. *P. micra* is anaerobic, asaccharolytic Gram-positive coccus found in dogs with periodontitis but not in the healthy dogs [68]. The virulence factors produced by *P. micra*, which may play a role in the pathogenesis of periodontitis, are poorly characterized. *P. micra* may modulate the inflammatory response in the host and contribute to the destruction of periodontal tissue. In addition, *P. micra* is capable of adhering to epithelial cells, also to other periodontal pathogens [69], and is able to form biofilms in conjunction with *Frederiksenia canicola* and *P. gulae*. *P. micra* might provide a catalyst for progressive tissue destruction, inflammation and alveolar bone loss in canine periodontal disease, in keeping with the keystone-pathogen hypothesis [68].

4.7 Genus *Prevotella*

The genus *Prevotella* is phylogenetically classified in the family *Prevotellaceae*, order *Bacteroidales*, class *Bacteroidia* and phylum *Bacteroidetes*. Representatives of this genus are Gram-negative, anaerobic, non-motile rods. The primary periodontal pathogen is species *Prevotella intermedia*. Within the *P. intermedia* strains, heterogeneity was found in terms of serology and DNA homology. In 1992, based on complex DNA-DNA hybridization, it was suggested that *P. intermedia* be reclassified into two species, *P. intermedia* and *P. nigrescens* [70]. *P. intermedia* and *P. nigrescens*, members of the "orange complex" described by Socransky et al., are among the most common species in subgingival plaque in humans. *P. intermedia* may under certain conditions increase the activity of degradation enzymes and promote the progression of periodontitis [71]. *P. intermedia* is also present in canine dental plaque. In dogs, the

counts of *P. intermedia* correlated with the amount of plaque and the degree of gingivitis [72]. *Prevotella dentalis* is also associated with periodontitis. *P. dentalis* (formerly *Mitsuokella dentalis*) was originally named after Japanese bacteriologist Mitsuok, who described this organism for the first time [38]. Mitsuoka isolates a large number of *P. dentalis* strains from humans, dogs and pigs that seem to be closely related to the *Bacteroides* genus [73].

4.8 Oral protozoa

For several decades, research in periodontology is focused on the characterization of bacterial communities thought to be involved in canine periodontal diseases. However, other microorganisms are known to inhabit the oral cavity and could also influence the process of periodontal disease. There were identified two oral protozoa, *Entamoeba gingivalis* and *Trichomonas tenax*, which can inhabit the canine oral periodontium. Both were statistically associated to animals with periodontal disease [74].

The species *Entamoeba gingivalis* is phylogenetically classified in the genus *Entamoeba*, class *Archamoebae* and phylum *Amoebozoa*. The protozoan *E. gingivalis* resides in the oral cavity and is frequently observed in the periodontal pockets of humans and pets. The parasite *E. gingivalis* is more prevalent and more abundant in periodontal pockets, suggesting that this ecological niche is either propitious for its survival, or that the parasite induces changes leading to this environment [75]. *E. gingivalis* is an opportunistic pathogen, which, together with synergistic symbiotic bacteria, can cause periodontal diseases in hosts with low immunity [76]. Pathogenicity of protozoa *E. gingivalis* in the oral cavity is not completely understood [77].

The species *Trichomonas tenax* is phylogenetically classified in the genus *Trichomonas*, family *Trichomonadidae* and order *Trichomonadida*. *T. tenax* inhabits the oral cavities of various mammals, including humans, dogs, cats and horses [78]. *T. tenax*, an anaerobic motile-flagellated protozoan, is 12–20 μm long and 5–6 μm wide organism. It is either ellipsoidal or ovoid in shape and has four anterior flagella of unequal lengths [79]. *T. tenax* can ingest bacteria and various particles by phagocytosis necessary for their development. *T. tenax*, detected in periodontal cases, is likely to be related to the onset and evolution of periodontal disease [80]. This parasite has been reported to be involved in a number of cases of pulmonary trichomoniasis. Besides bronchopulmonary exudates the trichomonads have also been found in pleural fluid, submaxillary gland and infra-auricular lymph node [81]. Several mechanisms may explain the deleterious effects of the *T. tenax* parasite towards periodontal tissues. Recent studies have emphasized the ability of parasites to induce changes in some features of microbial communities. *T. tenax* can escape the host immune response via a complex strategy caused by an imbalance of the oral cavity microbiocenosis. Pathogenic bacteria involved in periodontal host colonization and immune subversion use complement and toll-like receptor (TLR) signaling pathways. Like bacteria, parasites are recognized by TLR. *T. tenax* also produce fibronectin-like proteins, responsible for tissue adhesion. Given this pathogenic property, host-tissue disruption and lysis may be induced by *T. tenax* secretion of peptidases such as cathepsin B-like proteinases for matricial type 1 collagen and gelatin hydrolyses or haemolysins for erythrolysis [79].

5. The possibility of transferring bacteria from the oral microbiome of dogs to human

Except for to the impact on animal health, bacteria from the oral cavity of animals may also have harmful effects on human health in the case of microbial

transmissibility, for example, through dog bites. Dog bite wounds are polymicrobial, with a broad combination of aerobic and anaerobic microorganisms. The microbiology of infected bite wounds from dogs is similar to that of the organisms that colonize the dog's oral cavity. Less frequently, isolates may also come from the environment and patients' skin [82]. On average, a dog bite wound contains two to five different species of bacteria [83]. *Pasteurella* species are the most frequent isolates of dog bites (50%), especially *Pasteurella canis* is the most common isolate of dog bites [84]. Other common aerobic organisms include *P. multocida*, *P. dagmatis*, *Staphylococcus* spp. (including MRSA), *Streptococcus* spp. (including *S. pyogenes*), *Neisseria* spp., *Capnocytophaga canimorsus*, *Corynebacterium* spp., *Moraxella* spp., *Enterococcus* spp. and *Bacillus* spp. [82, 83]. The most common anaerobic organism isolated from infected dog bite wounds is *Fusobacterium nucleatum* [82]. *Fusobacterium canifelinum* was also isolated from wounds in humans after dog bites [85]. Other common anaerobes include *Prevotella* spp., *Bacteroides* spp., *Porphyromonas* spp., *Propionibacterium* spp. and *Peptostreptococcus* spp. [82, 83]. Several *Porphyromonas* species (*P. macacae*, *P. canoris*, *P. circumdentaria*, *P. cangingivalis*, and *P. cansulci*) [84] and other periodontal pathogens (*Tannerella forsythia*, *Prevotella intermedia* and *Prevotella dentalis*) were also isolated from infected dog bite wounds [82]. In addition, some pathogens such as *Leptospira*, *Rabies virus*, *Clostridium tetani* or *Francisella tularensis*, which can cause systemic infection after bites by dogs, were isolated from wounds in humans [83].

As transmission of oral bacteria during normal contacts between dogs and humans is also feasible one might expect correlations between the oral microbiota of dogs and humans [17]. Oral-to-oral transfer of *Neisseria shayeganii*, *Porphyromonas canigingivalis*, *Tannerella forsythia* and *Streptococcus* minor from dogs to humans is suspected. The finding of potentially zoonotic and periodontopathic bacteria in the canine oral microbiome may be a public health concern [15].

6. Conclusion

Review of literature showed that some bacterial species like *Tannerella forsythia*, *Campylobacter rectus*, *Treponema denticola*, *Fusobacterium nucleatum*, *Parvimonas micra* and *Prevotella intermedia* are the important pathogens for periodontitis in both humans and dogs. On the other hand, *Porphyromonas gulae* is specifically associated with canine periodontal disease. In addition, it is assumed, that oral protozoa such as *Entamoeba gingivalis* and *Trichomonas tenax* play role in canine periodontal disease. In summary, periodontal disease is polymicrobial disease and further analyses of the associated species of periodontitis and their virulence factors in dogs are needed.

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